COMPARISION OF EPIDURAL BUPIVACAINE AND BUPIVACAINE-MAGNESIUM SULPHATE COMBINATION IN LOWER ABDOMINAL SURGERIES

Dissertation submitted to
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
In partial fulfillment of the regulations
for the award of the degree of

M.D. BRANCH - X
ANAESTHESIOLOGY

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THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, INDIA
APRIL 2012

CERTIFICATE

This is to certify that the dissertation entitled "COMPARISION OF EPIDURAL BUPIVACAINE AND MAGNESIUM SULPHATE COMBINATION" is the bonafide original work of **Dr.P. LENIN** in partial fulfillment of the requirements for M.D. Branch-X (anaesthesiology) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in April 2012.

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DECLARATION

I **Dr. P. LENIN** solemnly declare that dissertation titled, **"**A COMPARISION OF EPIDURAL BUPIVACAINE AND BUPIVACAINE–MAGNESIUM SULPHATE COMBINATION is a bonafide work done by me at K.A.P.V. Government Medical College, during 2009-2012 under the guidance and supervision of my Chief **Prof. Dr. N. JOTHI, M.D.,D.A** Professor & Head of the department of Anaesthesiology.

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – X) in Anaesthesiology.

Place: Trichy

Date :

Dr. P. LENIN

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DR. P. LENIN

CONTENTS

INTRODUCTION

Epidural anesthesia was first performed by Spanish military surgeon **Fidel Pages** in 1921 in lumbar region. In 1949 **Curbelo** used **Tuohy** equipment for continuous blockade. The epidural technique became popular as it had some specific advantages over spinal anaesthesia. The feasibility of extended duration and differential blockade extended its application into other fields like post operative analgesia, chronic pain relief and obstetric pain relief. But some inherent negative points of epidural technique like delayed onset and patchy analgesia persist. Various attempts have been made to rectify these negative points.

Combined spinal-epidural technique got introduced in an attempt to rectify this and has become popular. Various additive drugs have been tried along with local anaesthetics in an attempt to hasten the blockade to improve the quality of block.

Among drugs that have been tried, magnesium sulphate deserves special mention. Even though magnesium sulphate has been used for various other purposes, its entry into anaesthetic arnamentorium is new. Magnesium sulphate has been described to possess various properties. The potentiation of local

anaesthetic drugs is one among them. So my study aims to evaluate the effect of its addition to local anaesthetic in epidural blockade.

Along with local anesthetic agent other additive drugs are Epinephrine, Clonidine , Dexmedetomidine, Neostigmine , Ketamine , and Benzodiazepines .

Magnesium sulphate a potent antagonist of NMDA receptors when used epidurally is claimed to hasten the onset of sensory blockade. Magnesium also causes physiological Calcium channel blockade and decreases the postoperative opioid requirements.

AIM OF THE STUDY

Sole epidural Bupivacaine is becoming unpopular due to delayed onset of sensory blockade. The aim of the study is to add magnesium sulphate as an adjunct to epidural Bupivacaine and evaluate

1. The impact of Magnesium sulphate on the quality of the block using the following parameters,

a. Onset of sensory blockade

b. Motor blockade quality

c. Two segment regression time

d. Post operative analgesia

2. Impact of Magnesium sulphate on hemodynamic changes.

EPIDURAL ANAESTHESIA

The term epidural analgesia mean a form of Regional Anaesthesia of injecting drugs through a catheter placed in the Epidural space.

The injection can cause both loss of sensation (anaesthesia) and a loss of pain (analgesia) by blocking the transmission of signal through nerve in or near the spinal cord.

HISTORY

In 1921 Spanish military Surgeon **Fidel Pages** uses the modern technique of Lumbar Epidural anaesthesia. Italian surgeons **Prof Achille Mario Dogliotti** popularized in 1930. **Dr. Robert A Hinson, Dr.Waldo B Edward and Dr. James L** South Worth working in Us Mariana Hospital developed the technique of continuous caudal anaesthesia. In 1949 **Curbelo** used **Touhy** equipment for continuous blockade.

LOCATION:

Between the spinal Dura and the Spinal periosteum lies the Epidural Space. It extends from the Foramen magnum to Sacrococcygeal membrane.

CONTENTS:

It contains Nerve roots, Fat, Blood vessels, lymphatics and valveless venous plexus of Baston.

BOUNDARIES:

Posterior: Laminae and Ligamentum flavum.

Sides : Pedicles of the vertebral body and the intervertebral spaces.

Anterior : Bodies of the vertebrae, intervertebral discs and Posterior longitudinal ligament.

Thickness of Ligamentum Flavum:

Cervical : 1.5-3.0 mm

Thoracic : 3.0-5.0 mm

Lumbar : 5.0-6.0 mm

Caudal : 2.0-6.0 mm

Distance from the skin is about 4-6cm in 80% of the individuals. The Spinal canal is roughly triangular in cross section and therefore the space is deepest in the midline posterior. The Epidural space is larger in the caudal than the cephalic segment.

Each spinal nerve as it passes through its intervertebral foramen into the paravertebral space carries with it a collar of the fatty aerolar tissue of the epidural space. Paravertebral space both serial and controlateraly communicate with each other through the epidural space.

There is a negative pressure in the epidural space. The proposed reasons for the presence of negative pressure are: The natural effects of Starling forces across capillary walls produce a low fluid pressure in all tissues on the basis of oncotic pressure, these results in sub-atmospheric pressure and tissue collapse in the spaces of the opposing surfaces of the spinal canal.

Dural tenting during needle advancement may also contribute to sub-atmospheric pressure.

Sub-atmospheric intrathoracic pressure is thought to contribute to the occurrence of sub-atmospheric pressure in the thoracic epidural level.

VEINS: The Epidural space contains a network of veins there run mainly in a vertical direction and form 4 main trunks. These trunks communicate freely by venous rings at each vertebral level.

The epidural veins are valveless and form a connecting link both between the pelvic veins below and the cerebral veins below and the cerebral veins above, a possible pathway for the spread of bacteria and malignant cells.

The increase in CSF pressure that accompanies laughing and straining results in part from the shunt of blood from thoracic and abdomen veins into the thin walled vertebral veins. The veins of the epidural space will therefore be distended if thoracic or abdomen pressure is increased thus having bloody tap.

ARTERIES: The arteries of the epidural space are relatively insignificant and originate from the arteries corresponding to the named veins. The arteries enter at each intervertebral foramen lie chiefly in the lateral part of the epidural space and supply the adjacent vertebrae, ligament and spinal cord.

PHYSIOLOGY:

After epidural injection the local anaesthetic drug binds to the spinal nerve in the epidural spaces, spinal nerve rootlets within the CSF and within the spinal cord.

It enters into the CSF by means of Dural sleeves and spinal radicular arteries. Some of the drug goes into the paravertebral space and block the nerves.

RESPIRATORY PHYSIOLOGY: At the midthoracic level of blockade Pulmonary function test, Gas exchange and control of breathing are generally preserved in patients without preexisting respiratory diseases.

Subjective sensation of dyspnea is due to decreased sensation of expansion of chest wall with inspiration. However Tidal volume, respiratory rate, minute ventilation and lung volumes are maintained in healthy resting patients. Small block height will affect accessory respiratory muscle (abdomen and intercostals) which plays major role in expiration which in turn reduces peak expiratory flow.

CARDIAC PHYSIOLOGY: Epidural blockade results in a lesser degree of sympathetic block and much CVS stability than Subarchanoid block. Level of sensory block is the same as sympathetic block in Epidural.

Blockade below T4: Results in low thoracic and lumbar will cause in a peripheral sympathetic blockade with vascular dilation in the pelvis and lower limb, then pooling of blood in the abdomen viscera. This will lead to reduced venous return thereby reduced cardiac output. Increased activity of cardiac sympathetic fibers T1-T4 result in increased cardiac contraction and heart rate. This will maintain normal cardiac output.

Blockade above T4: Will cause decreased Heart rate and contraction by acting at cardiac sympathetic fibers T1-T4 will leads to bradycardia and hypotension.

GASTROINTESTINAL, HEPATIC AND GENITOURINARY PHYSIOLOGY:

The sympathectomy of epidural anaesthesia results in relaxation of sphincters, contraction of bowels and increased secretion caused by parasympathetic predominance. Hepatic blood flow is related to mean arterial pressure and thus maintained if the patient in hemodynamicaly stable. Likewise renal blood flow and perfusion is preserved.

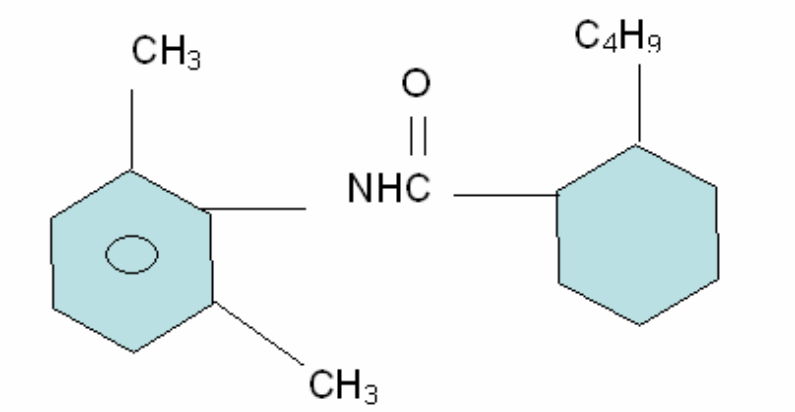
BLADDER: Temporary atonia in lumbar epidural block is due to block of S2-S4. In continuous epidural blockade bladder to be catheterisied.

HYPOTHERMIA: Common in epidural block is due to heat loss to the cold environment due to sympathectomy induced vasodilatation.

NEURO ENDOCRINE EFFECT OF EPIDURAL ANAESTHESIA:

Surgical stress is associated with a variety of changes in Endocrine and metabolic function including Protein metabolism leads to negative nitrogen balance. This stress is prevented by appropriate sensory blockade.

PHARMACOLOGY OF BUPIVACAINE



Bupivacaine

It is an amide local anaesthetic characterized as Pipecoloxylidides. Addition of a butyl group to the piperidine nitrogen of mepivacaine results in Bupivacaine. It is a chiral drug because of possession of asymmetric carbon atom.

It was first synthesized in Swedon by **Ekenstam a**nd his colleagues in 1957 and used clinically by **L.J.Telivuo** in 1963. Its molecular weight is 288.

Mechanism of Action:

It prevents transmission of nerve impulses by inhibiting passage of sodium ions through ion selective sodium channels in nerve membranes. They do not alter the transmembrane potential or threshold potential.

PHARMACOKINETICS:

It is a weak base that has pka value above physiologic pH 7.4 only 15% exists injection, dosage and use of epinephrine. Lung is capable of extracting bupivacaine from circulation, which will in nonionised form. Absorption depends on the site of limit concentration of drug that reaches systemic circulation. This first pass pulmonary extraction is dose dependant suggesting that it becomes saturated rapidly.

Pka	:	8.1
Protein Binding	:	95%
Lipid Solubility	:	28
Volume of distribution	:	73 litre
Clearance of drug from plasma	:	0.417 lit/min
Elimination half life	:	210 min
Onset time	:	10-15 min

METABOLISM

Slowest metabolism among amide local anaesthetics. It undergoes aromatic hydroxylation, N-dealkylation, amide hydrolysis and conjugation. Only the N-desbutyl bupivacaine has been measured in blood or urine after epidural or spinal anaesthesia. Alpha-1 acid glycoprotein is the most important protein binding site of bupivacaine.

SIDE EFFECTS

Bupivacaine is more cardio toxic than equieffective doses of lignocaine. This is manifested by severe ventricular arrhythmias and myocardial depression. Bupivacaine blocks cardiac Na+ channels rapidly during systole and dissociates more slowly during diastole, so that a significant fraction of Na+ channels remain blocked at the end of the diastole. Thus the block by Bupivacaine is cumulative and substantially greater.

CLINICAL USE

Onset of anaesthesia and duration of action are long. Its tendency to provide more sensory than motor block has made it popular for providing postoperative analgesia. Used mainly for

Epidural anaesthesia

Spinal anaesthesia

Infiltration anaesthesia

Field block anaesthesia

Nerve block anaesthesia

PHARMACOLOGY OF MAGNESIUM

Magnesium is a bivalent ion with an atomic weight of 24.312. Human body contains 1 mole (24g) of magnesium. It is the fourth common mineral salt in the body after phosphorus, calcium and potassium, second intracellular cation after potassium. In serum Magnesium is divided into three fractions:

1) Ionized

2) Protein bound and

3) Contained in anion complexes

These fractions account for 65%, 27% and 8% in serum concentration respectively.

CHEMICAL STRUCTURE OF MAGNESIUM SULPHATE

PROPERTIES OF MAGNESIUM SULPHATE:

1) CELLULAR PROPERTIES:

Magnesium intervenes in the activation of membrane calcium ATPase and Na+K+ATPase involved in transmembrane ion exchange during depolarization and repolarization phases. It acts as a stabilizer of cell membrane and intracytoplasmic orgenelles.

2) **ION CHANNELS**:

It acts as a regulator of different ion channels. It has a competitive antagonist action against calcium inflows thereby limiting the outflow of calcium from sarcoplasmic reticulum. So it is a calcium blocker and calcium channel modulator.

3) CARDIOVASCULAR SYSTEM:

It acts on calcium channels in the myocardial muscle and also acts indirectly on the cardiac muscle by inhibiting the calcium uptake on the Troponin C of the myocytes and thereby influencing myocardial contractility.

Its vasodilatory action is due to its activation of cyclic AMP. This causes reduction in systolic blood pressure.

Pulmonary vascular resistance is unaltered.

Coronary vascular resistance is reduced and causes vasodilation.

4) **NEUROMUSCULAR TRANSMISSION**:

It has preponderant presynaptic and postsynaptic effects. Magnesium acts competitively in blocking the entry of calcium into the presynaptic release of acetylcholine is reduced by magnesium, thereby decreasing the effect of acetylcholine on the postsynaptic receptors, which in turn increases the threshold of axonal excitation.

It also produces progressive inhibition of catecholamine release from the adrenal medulla, adrenergic nerve endings and adrenergic post ganglionic sympathetic fibers.

5) **RESPIRATORY SYSTEM**:

It has bronchodilatory action due to the inhibition of smooth muscle contraction, histamine release from the mast cells and acetylcholine release from the cholinergic nerve endings.

6) Magnesium is involved in hundreds of enzyme reactions in the body.

7) Acts as antagonist of **NMDA** receptors and this explains its use in post-operative analgesia.
8) Magnesium sulphate increases the production of prostaglandins causing vasodilatation of the small intracranial vessels which is responsible for its anticonvulsant action.

CLINICAL USES:

1) **For Severe Preeclampsia and Eclampsia:**

A loading dose of 4-6gm magnesium sulphate diluted in 100ml of normal saline given over 15min intravenously. Then 2gm/hr in 100ml of IV infusion (maintain serum levels between4 and 7mEq/L).

Intermittent injection:

4gm given slow IV followed by 10gm,5gm in each buttocks as deep IM injection. Then every 4hours 5gm intramuscularly upto 24hours after delivery.

2) Magnesium sulphate has a tocolytic effect at serum levels of 8-10mEq/L. Loading dose of 4-6gm over 20min intravenously, then after the contraction

ceases maintenance is done using 2-4gm per hour intravenously for 12-24 hours.

3) To reduce the stress response during intubation, magnesium sulphate is used in the dosage of 30-50mg/kg. intravenously.

4) In surgery for Phaeochromocytoma it helps to maintain haemodynamic balance because it inhibits the catecholamine release from adrenal medulla and adrenergic nerve endings.

5) Nephritic Seizures: In children with nephritic seizures, the 50% concentration should be diluted to a 20% solution for i.m. injection. The dose for children is 20 to 40 mg (0.1 to 0.2 ml of 20% solution)/kg of body weight, administered i.m. as needed, to control seizures.

6) It is used postoperatively in patients who have undergone Coronary artery bypass grafting to reduce the incidence of ventricular arrhythmias.

7) It is also used in the treatment of Torsades De Pointes, as intravenously or intraosseously in the dosage of 25 to 50 mg/kg (upto 2gm).

8) **Acute myocardial infarction**: Magnesium sulphate is used is used in the dose of 2gm intravenously over 5-15 min followed by 18 gm over 24hrs as infusion.

9) **Total Parenteral Nutrition**: In total parenteral nutrition, maintenance requirements for magnesium are not precisely known. The maintenance dose

recommended for adults is 5 to 8 mEq magnesium/L of TPN solution; typical daily intake ranges from 0.25 to 0.6mEq/kg/day for infants. For adult ranges from 10 to 24 mEq.

10) In barium poisoning: 1-2gm is used to counteract the intense muscle stimulating effects of barium.

11) In refractory bronchial asthma it is used for its bronchodilatory action.

12) **Hypomagnesemia :** in case of mild deficiency 1 gm every 6hours for 4 doses, in severe cases 1-5gms (2- 10ml of 50% solution) in divided doses, repeated until the serum levels are normal.

13) Recent studies show its use in Tetanus patients, at a serum concentration of 2-4mEq/L, it gives good control of spasms and muscle rigidity.

14) Magnesium sulphate is used in the dose of 50 mg intrathecally for potentiation of opioid analgesia.

PRECAUTIONS:

Because magnesium is removed from the body solely by the kidneys, the drug should be used with caution in patients with renal impairment. Urine output should be maintained at a level of 25-50 ml per hour. Monitoring serum

magnesium levels and the patient's clinical status is essential to avoid the consequences of over dosage in toxemia. Clinical indications of a safe dosage regimen include the presence of the patellar reflex (knee jerk) and absence of respiratory depression (approximately 16 breaths or more/minute). Serum magnesium levels usually sufficient to control convulsions range from 3 to 6 mg/100 ml (2.5 to 5.0 mEq/L). The strength of the deep tendon reflexes begins to diminish when magnesium levels exceed 4 mEq/L. Reflexes may be absent at 10 mEq magnesium/L, where respiratory paralysis is a potential hazard. An injectable calcium salt should be immediately available to counteract the potential hazards of magnesium intoxication in eclampsia.

PREPARATIONS AVAILABLE:

Parenteral injection: Magnesium sulphate-	10%, 12.5%, 50%
For Intravenous use only -	4%, 8%.
Magnesium sulphate in dextrose :	1% in 5% dextrose.
	2% in 5% dextrose.

When administered intravenously the onset of action is immediate and duration of action is 30 min. on administration by intramuscular route the onset of action takes 1hr and duration of action is 3-4 hrs.

Storage: 15-30degree centigrade. For IV use concentration of 20%or less should be used. Rate of injection should be 1.5ml/hr.

DRUG INTERACTIONS:

Central nervous system depressants: When barbiturates, opiates, general anaesthetics, or other CNS depressants are administered concomitantly with magnesium sulphate, dosage of these agents must be carefully adjusted because of the additive central depressant effects.

Neuromuscular blocking agents: Excessive neuromuscular blockade has occurred in patients receiving parenteral magnesium sulfate and a neuromuscular blocking agent; these drugs should be administered concomitantly only with caution.

Cardiac glycosides: Magnesium salts should be administered with extreme caution in digitalized patients, because serious changes in cardiac conduction, which can result in heart block, may occur if administration of calcium is required to treat magnesium toxicity.

ADVERSE REACTIONS:

The adverse effects of parenteraly administered magnesium usually are the result of magnesium intoxication. These include flushing, sweating, hypotension, depressed reflexes, flaccid paralysis, hypothermia, circulatory collapse, cardiac and CNS depression proceeding to respiratory paralysis. Hypocalcaemia with signs of tetany secondary to magnesium sulphate therapy for eclampsia, has been reported.

SYMPTOMS AND TREATMENT OF OVERDOSE:

Magnesium intoxication is manifested by a sharp drop in blood pressure and respiratory paralysis. Disappearance of the patellar reflex is a useful clinical sign to detect the onset of magnesium intoxication. In the event of over dosage, artificial ventilation must be provided until a calcium salt can be injected i.v. to antagonize the effects of magnesium.

In adults i.v. administration of 5 to 10 mEq of 10% of calcium gluconate will usually reverse respiratory depression or heart block due to magnesium intoxication. In extreme cases, peritoneal dialysis or hemodialysis may be required.

Hypermagnesemia in the newborn may require resuscitation and assisted ventilation via endotracheal intubation or intermittent positive pressure ventilation, as well as i.v. calcium.

NMDA RECEPTORS

The NMDA receptors is a glutamate activated calcium ionophore that is composed of a series of subunits.

NMDA – mediated behavior: Blockade of the spinal NMDA receptors by intrathecal delivery does not alter acute thermal or mechanical threshold. This receptors does play an important role in augmenting afferent evoked excitation in the face of conditioning stimulation.

PHYSIOLOGICAL EFFECTS:

Activation of afferent NMDA receptors will initiate the release of substance P while blockade of NMDA receptors not AMPA receptors will significantly diminish substance P release evoked from small primary afferents. This activation by NMDA receptors may reflect both the depolarization of the terminal by the ionophore activation. However in the spinal dorsal horn, in the absence of conditioning stimulation, the NMDA receptors fail to be functional in the presence of Glutamate. This lack of activation reflects at least in the presence of Magnesium ions that occupies and occludes the pore at resting membrane potentials. In the face of persistent depolarization of the membrane, as with frequent stimulation of C fibers, the membrane is adequately depolarized the Magnesium block is removed and the channel become functional passing large amounts of calcium and associated curre

MATERIALS AND METHODS

After approval of the study by our institutional ethics committee, the study was conducted on 50 ASA grade I or II patients undergoing elective lower abdominal surgeries. Lumbar epidural anesthesia was performed to all the patients. The age of the patients ranged from 23- 70 weighing 45-80 kg and height ranging from 150 – 172 cm. all patients were thoroughly examines preoperatively. Informed consent was obtained from all of them.

In the assessment room, vital parameters like pulse , blood pressure, and base line investigations like hemoglobin, urine analysis for albumin and sugar, blood sugar ,urea and creatinine and Electrocardiogram were checked. Thorough examination of all the systems and airway assessment was done.

Exclusion criteria including significant co-existing diseases , long term analgesic use, and contraindications to regional anaesthesia such as local infection and bleeding diathesis.

The patients were randomly allotted to 2 groups each containing 25.

Group C:

Patients received 19ml of 0.5 % Bupivacaine + 1 ml normal saline

Group S:

Patients received 19ml of 0.5 % Bupivacaine + 50 mg of magnesium sulphate at L2-L3 space using 17G Tuohy needle and placing epidural catheter at 8 cm.

The total volume of the injecting solution was 20 ml in both groups. In the operating theatre Boyles apparatus emergency drugs and airway devices were kept ready. Patients were shifted to operating table. Non invasive blood pressure and Electrocardiogram leads were connected to the patient. Preoperative baseline systolic and diastolic blood pressure, pulse rate, oxygen saturation were recorded. Patients were cannulated with 18G intravenous needle and preloaded with 1 litre of Ringer Lactate. The patient was placed in right lateral position. The skin over the back was prepared with antiseptic solution and draped with sterile towel. After infiltrating skin and subcutaneous tissue with local anaesthetic , 17 G Tuohy needle inserted at L2-L3 space and epidural catheter inserted and placed in 8 cm. after giving test dose of 2% xylocaine with 1:200000 dilution adrenaline were given. After checking any change in pulse rate and able to dorsiflex the great toe,

the position of the catheter was confirmed. After 20 minutes the prepared solution was injected. Then the patient was made to lie down immediately and the time of injection of epidural anesthetic was noted.

SENSORY BLOCK:

The onset of sensory block was defined as the time between the injection of anaesthetic solution and the absence of pain at L1 dermatome level. Sensory block was assessed by loss of sensation by pinprick at L1 level. This pinpricking continued till the peak block height was reached and the time was noted. The duration of sensory block was defined as the time for regression of two segments from the maximum block height evaluate by pinprick. Sensory block was checked every 10 mins till it reaches two segment regression levels.

MOTOR BLOCK:

Motor block was assessed bilaterally using the Dorsiflexion of the ankle

Assessment of motor block was started immediately after turning the patient supine and continued every minute till the patient is unable to dorsiflex the ankle joint.

VITAL SIGNS AND SIDE EFFECTS:

Vital parameters like systolic and diastolic blood pressure, pulse rate and oxygen saturation were recorded every 2mins for the first 10min and thereafter

every 5 mins until the immediate postoperatively period. Hypotension was defined as fall in systolic blood pressure more than 30% from the baseline of systolic blood pressure less than 90 mmHg. This was managed with intravenous ephedrine in incremental dose of 6 mg.

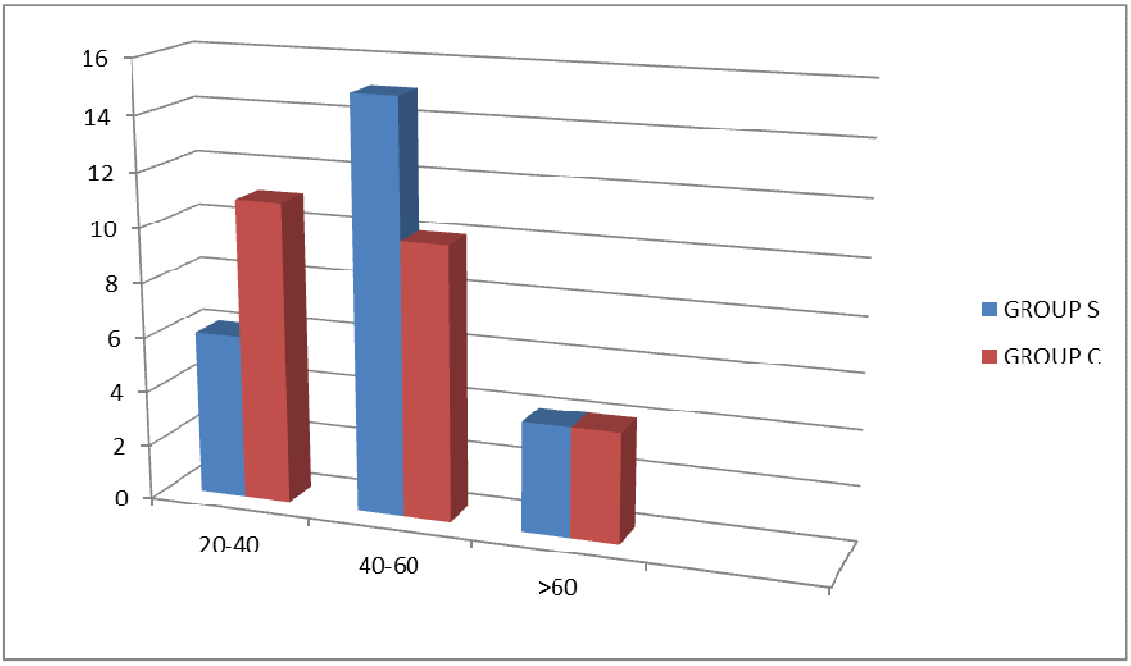
Bradycardia was defined as heart rate of less than 60/min. and was planned to be managed with IV atropine in incremental doses. Respiratory depression was said to be present if respiratory rate was less than 8/min or Oxygen

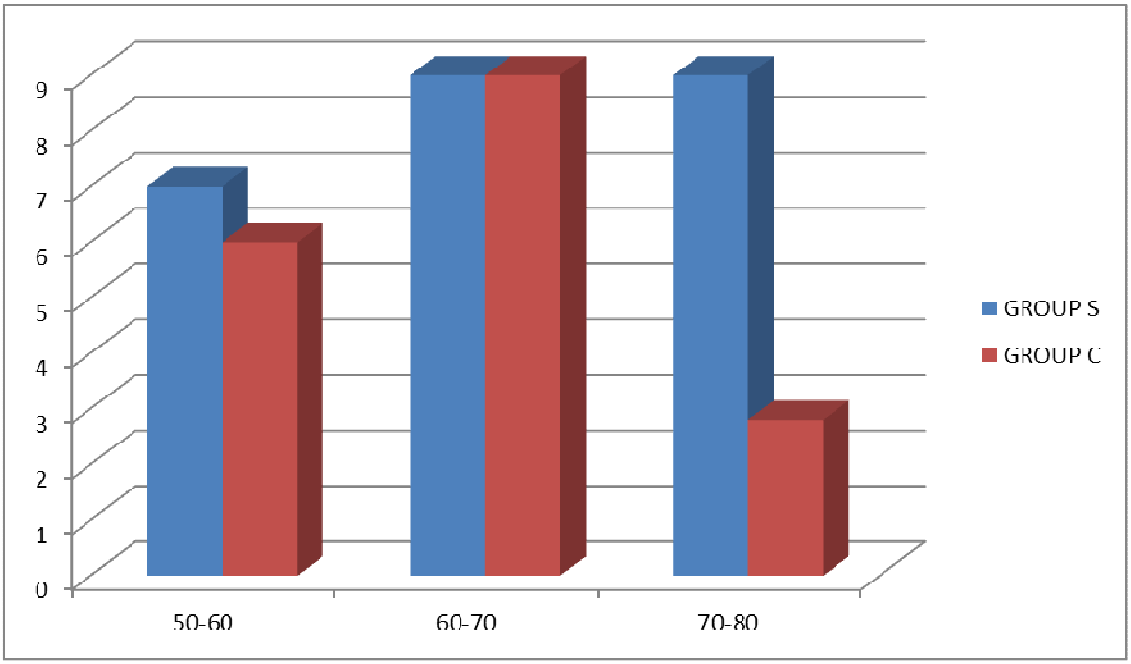
saturation less than 85%. This was planned to be managed with mask ventilation. Vomiting if present was planned to be managed with Inj.Ondensetron 8mg intravenously. Pruritis was planned to be managed with reassurance or Inj. Pheniramine maleate 22.75mg intravenous. Patients were shifted to post anesthesia care unit after completion of surgery. Vital signs were recorded every 15 min in the first hour after surgery, 30min for the next 2hrs. And there after every hour for the next 3 hours. Patients were shifted to post operative ward after complete resolution of motor blockade and stabilization of blood pressure

DURATION OF ANALGESIA:

The time at which the patient complained pain was noted. The duration of effective analgesia was defined as the period from the epidural injection to the first occasion when the patient complained of pain in the postoperative period.

OBSERVATION AND ANALYSIS





OBSERVATION AND ANALYSIS

Of the fifty patients involved, 25 belonged to group C and other 25 belonged to group S

AGE DISTRIBUTION

The age distribution in group S 23-73. Age distribution in control group 27-70.the mean age and age distribution was similar stastically.

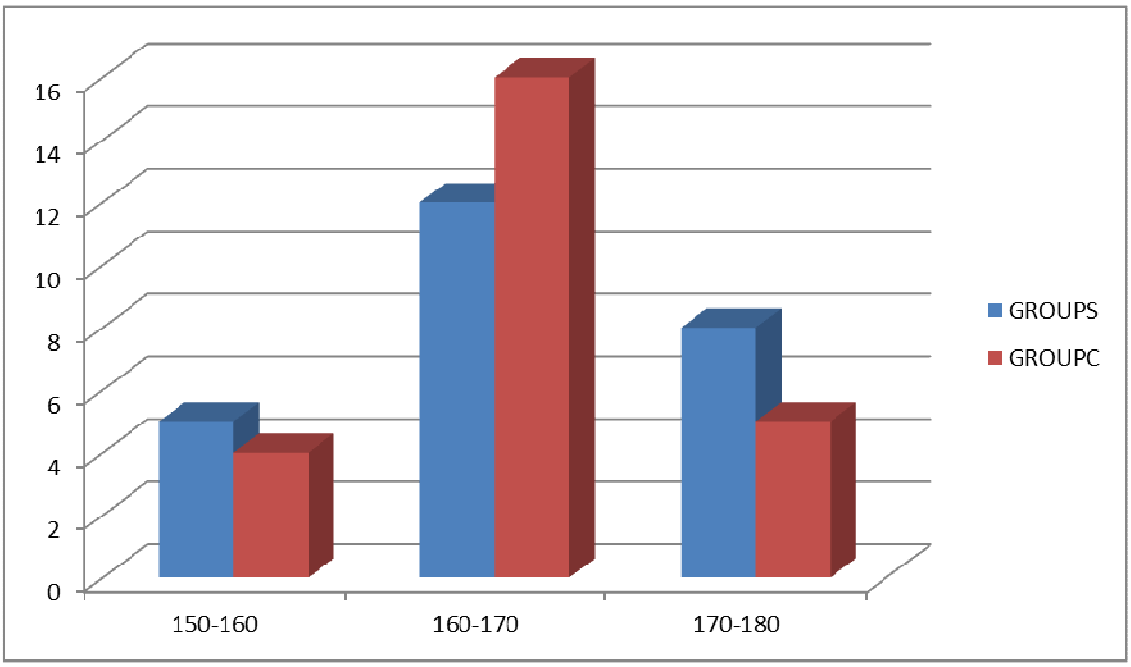
AGE IN YEARS	GROUP S	GROUP C
20-40	23	27
MAXIMUM	73	70
MEAN	48.33	44.6

Weight distribution

Weight distribution in group S 54-80, while in control group 52-72. Mean weight of the patients were comparable.

Weight in kg	Group s	Group c
Range	54-80	52-72
mean	66.32	65.52

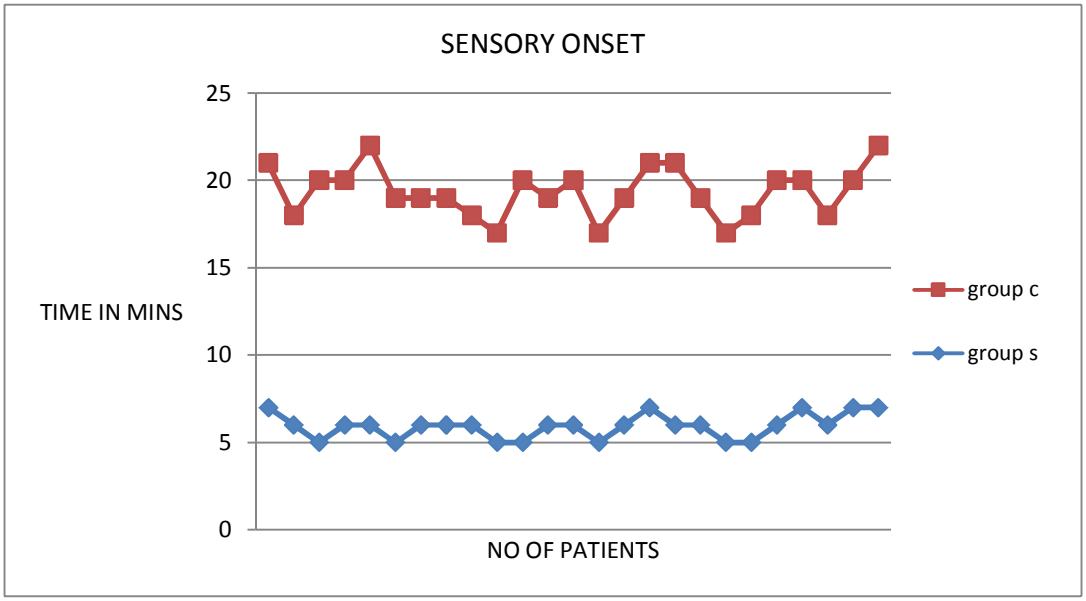
Height distribution

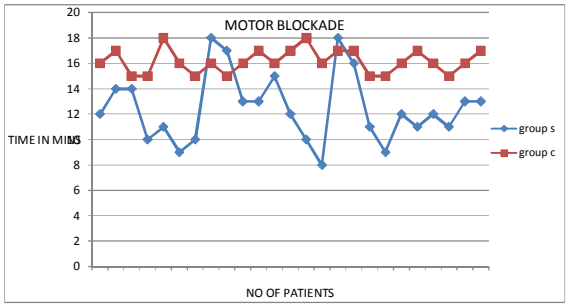


Height distribution

Height distribution in group S 156-172 cms, while in control group 153-172. Mean height in both group patients were comparable.

Height in cm	Group S	Group C
range	156-172	153-172
mean	164.36	165.08


SENSORY ONSET
25
20
15
10
5
0
TIME IN MINS
NO OF PATIENTS
group c
group s


MOTOR BLOCKADE
20
18
16
14
12
10
8
6
4
2
0
TIME IN MINS
NO OF PATIENTS
group s
group c

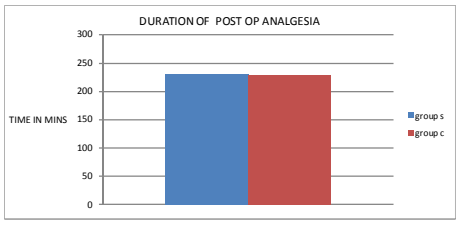
Sensory blockade

Sensory blockade min	Group C	Group S	P value
Onset	13.44± 1.16	5.92±0.70	<0.0001

Motor blockade

Motor blockade	Group S	Group C	P value
Onset	12.48± 2.67	16.16± 0.94	<0.0001


TWO SEGMENT REGRESSION TIME
TIME IN MINS
160
140
120
100
80
60
40
20
0
145.36
136.96
group s
group c

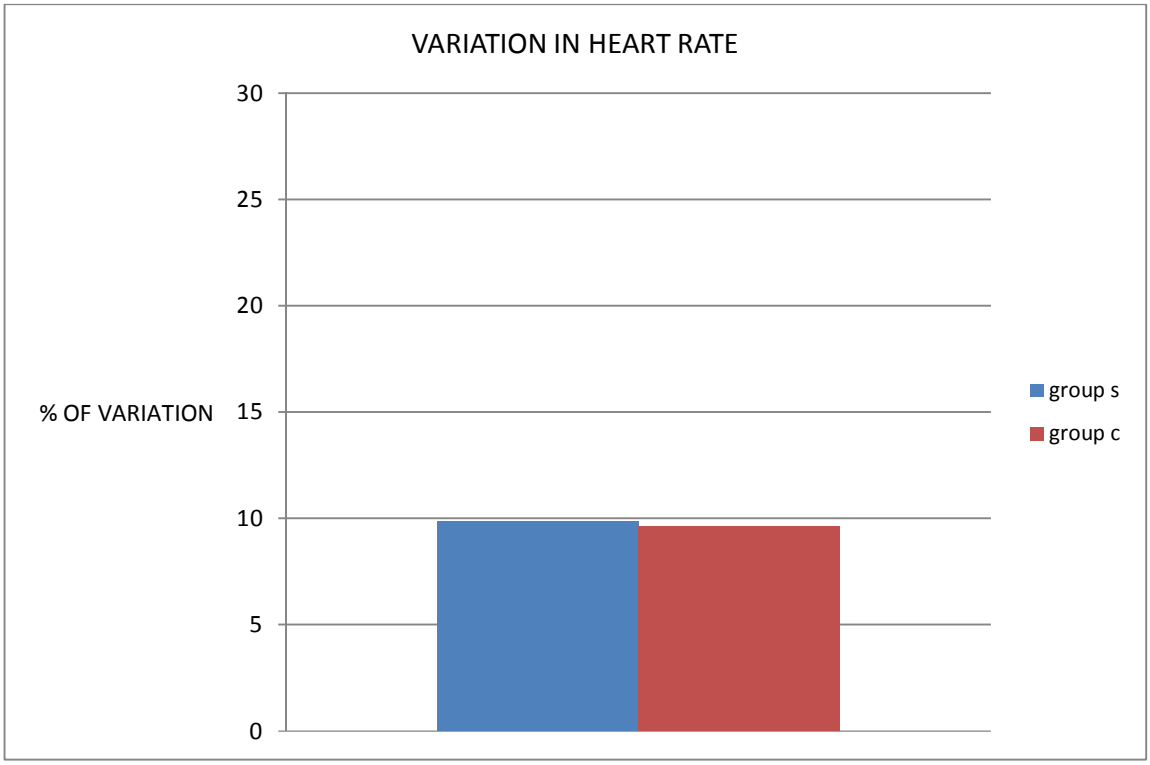

DURATION OF POST OP ANALGESIA
TIME IN MINS
300
250
200
150
100
50
0
group s
group c

TWO SEGMENT REGRESSION TIME

TWO SEGMENT REGRESSION TIME MIN	GROUP S	GROUP C
MEAN ± SD	145.36±4.97	136.96±8.19

DURATION OF POST-OP ANALGESIA

DURATION OF POST-OP ANALGESIA MINS	GROUP S	GROUP C
MEAN ± SD	231.04±12.63	228.48±8.81


VARIATION IN HEART RATE
% OF VARIATION
30
25
20
15
10
5
0
group s
group c

Percentage of heart rate changes(below the base line level)

	Group s	Group c
Heart rate changes % (mean)	9.84	9.64

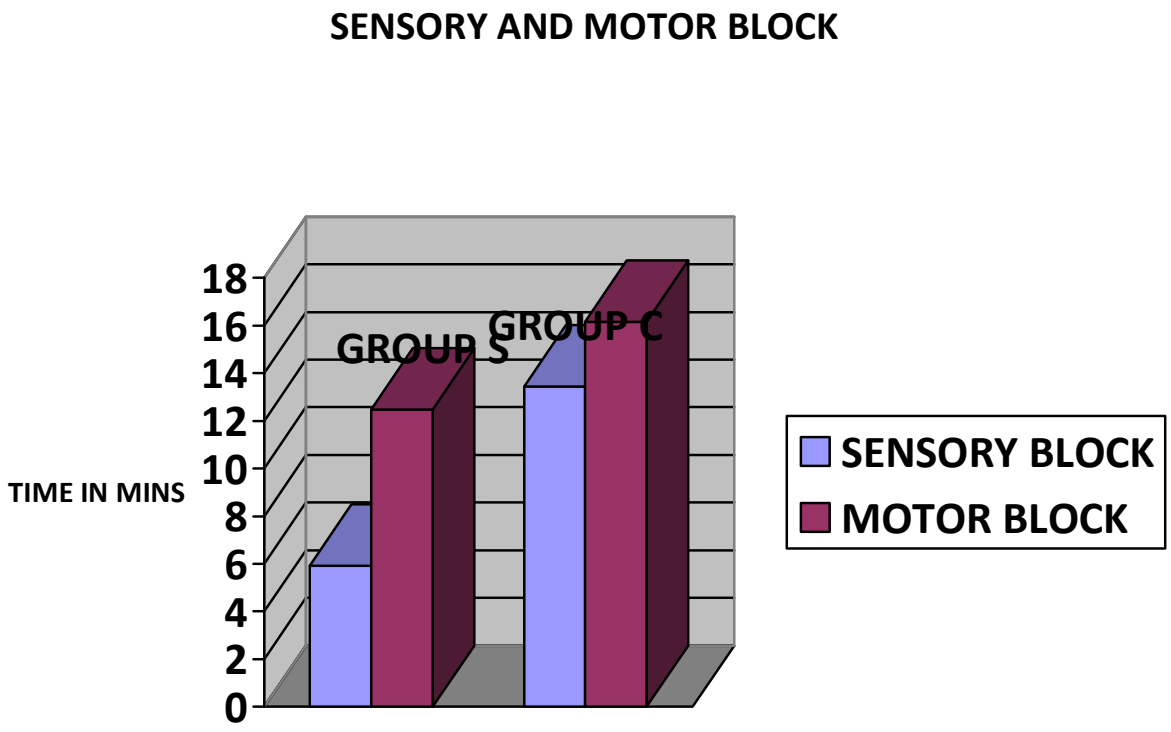
PARAMETERS	GROUP S	GROUP C	P VALUE
AGE	48.3± 13.00	44.6±13.213	0.3233
HEIGHT	165.08±5.445	164.36±5.345	0.639
WEIGHT	66.32±7.767	65.52±7.428	0.7148
ONSET OF SENSORY BLOCK	5.92±0.702	13.44±1.158	<0.0001
ONSET OF MOTOR BLOCK	12.48± 2.650	16.16±0.943	<0.0001
TWO SEGMENT REGRESSION TIME	145.36±4.97	136.96±8.19	<0.0001
DURATION OF POST OP ANALGESIA	231.04±12.633	228.48±8.813	0.41

DISCUSSION

The primary aim of this study was to evaluate the effect of adding magnesium sulphate to Bupivacaine in Epidural anaesthesia. The safety of Epidural magnesium sulphate administred in humans and animals have been established. Simpson and Kroin demonstrated in animals that epidural magnesium sulphate has a safety profile.

The dose of magnesium sulphate used in this study was based on data Tammoy Ghatak and Girish Chandra where 50 mg of magnesium sulphate added to epidural Bupivacaine. In their study magnesium sulphate has quickens the onset of sensory blockade. The duration of post-operative analgesia are also increased.

SENSORY AND MOTOR BLOCKADE

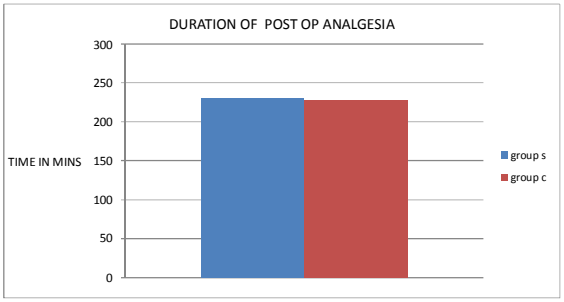


PARAMETERS	GROUP S	GROUP C	P VALUE
ONSET OF SENSORY BLOCK	5.92±0.702	13.44±1.158	<0.0001
ONSET OF MOTOR BLOCK	12.48± 2.650	16.16±0.943	<0.0001

In the study group the time of onset of sensory blockade was 5.92±0.702 mins, whereas in control group was 13.44±1.158(p value <0.0001) which shows that there is a significant difference in the onset time. The addition of magnesium sulphate has definitely decrease the sensory onset time.

The time of onset of Motor blockade in study group was 12.48±2.670 whereas in the control group Motor blockade was 16.16±0.943(p value <0.0001) which shows that the difference is statistically significant.

TWO SEGMENT REGRESSION TIME



The mean duration of two segment regression time in study was 145.36±4.974, whereas in control group was 136.96±8.19 (p value <0.0001).

TWO SEGMENT REGRESSION TIME

TWO SEGMENT REGRESSION TIME MIN	GROUP S	GROUP C	P VALUE
MEAN ± SD	145.36±4.97	136.96±8.19	<0.0001

DURATION OF POST-OPERATIVE ANALGESIA

DURATION OF POST OP ANALGESIA MINS	GROUP S	GROUP C	P VALUE
MEAN ± SD	231.04±12.63	228.48±8.81	0.4033

The duration of Post-operative analgesia was 231.40±12.633 mins in study group and 228.48±8.813 in control group (pvalue <0.41) which shows 2 groups

were not statistically significant. The probability value was detected by unpaired two sample student 't' test.

This implies that additive of magnesium sulphate to epidural Bupivacaine will quickens the onset of sensory blockade with minimal prolongation of post-operative analgesia.

This correlate the study of T.Ghatak and G. Chandra of addition of epidural magnesium sulphate to bupivacaine to reduce the time of onset of sensory blockade.

OTHER PARAMETERS

The change in Heart rate was 9.84% in study group whereas in control group 9.64% which shows there is no significant change. The usage of Ephedrine was also there is no significant changes.

SUMMARY

We conducted a double blinded randomized controlled study in 50 patients belonging to ASA I and II undergoing elective lower abdominal surgeries to evaluate the effect of adding magnesium sulphate to bupivacaine and bupivacaine alone in epidural anaesthesia in KAPV Govt Medical college hospital. For the same reason, we divided randomly the patients into two groups of 25 each.

Group C received 19 ml of 0.5% of bupivacaine (95mg) and 1ml of Normal saline.

Group S received 19 ml of 0.5% of bupivacaine (95 mg) and 1ml of Magnesium sulphate (50 mg).

The total volume of the injected solution was 20 ml in both groups. The onset of sensory and motor blockade, the duration of post-operative analgesia were noted in the both the groups . Demographic data were similar in both the groups.

We found the onset of sensory blockade was faster in the magnesium group.

The duration of post-operative analgesia was slightly prolonged in the study group.

The incidence of side effects were similar in both the groups.

There is no difference in the change in heart rate and the episode of hypotension were also similar in both the groups.

CONCLUSION

This study concludes that epidural magnesium sulphate when added to bupivacaine will shorten the onset of sensory blockade significantly in patients undergoing elective lower abdominal surgeries without increasing the incidence of side effects.

REVIEW OF LITERATURE

Tanmoy Ghatak, Girish Chandra, Anita Malik has done a study about the effect of adding epidural magnesium sulphate and clonidine to bupivacaine in patients undergoing lower abdomen surgeries. A total of 90 ASA grade I and II patients undergoing lower abdomen surgeries were enrolled to receive either magnesium sulphate (group B) or Clonidine (Group C) along with epidural bupivacaine for surgical anesthesia. All patients received 19ml of epidural bupivacaine 0.5% along with 50 mg magnesium in group B, 150mcg clonidine in group C, whereas in control group (Group A) received same volume of normal saline. The onset of sensory blockade was quicker in magnesium (Group B). In group C there was prolongation of duration of anaesthesia and sedation with lower VAS score. The study explained the probable reason for quicker onset sensory blockade in magnesium group is it is a NMDA receptor antagonist. Magnesium also has the property of anti noiciceptive effect in animal models. There are no additional side effects in both groups.

A A Yousef and Y M Amr has demonstrated the effect of adding magnesium sulphate to epidural bupivacaine and fentanyl in elective caesarean section using combined spinal-epidural anaesthesia. Patients were allocated in two groups of ASA I or II. All received 2ml intrathecal 0.5% hyperbaric bupivacaine, 10 ml

epidural 0.25% plain bupivacaine with fentanyl 100mcg and the other receive 10 ml epidural 5% magnesium sulphate. The group received magnesium has faster onset of sensory block and greater motor block and muscle relaxation ($p<0.05$) and also later onset of post operative pain. There is no difference in hypotension, nausea and vomiting.

R.Arcioni and S. Palmisami has conducted a study of giving intrathecal and epidural magnesium sulphate supplementation of spinal anaesthesia to patients undergoing orthopedic surgeries. Patients were randomly assigned to receive intrathecal magnesium sulphate(94.5mg 6.3%) epidural magnesium sulphate (2% 100mg/hr) intrathecal and epidural magnesium sulphate combined or spinal anaesthesia (L bupivacaine and sufentanyl) alone . In patients receiving spinal anaesthesia with combined intrathecal and epidural magnesium sulphate significantly reduces post-operative analgesia requirement. Magnesium sulphate alters pain processing and reduces the induction and maintenance of central sensitization by blocking the NMDA receptor in the spinal cord.

A. Bilir and S. Gulec has conducted with 50 patients undergoing hip surgery were enrolled to received either fentanyl(F) or Fentanyl plus magnesium sulphate (FM) for 24 hour for epidural analgesia. All patients were equipped with a patient

controlled epidural analgesia device and the initial setting of a demand bolus of Fentanyl 25mcg, group FM received 50 mg magnesium sulphate epiduraly as an initial bolus dose followed by a continuous 100 mg day. Since Magnesium has antinoiciceptive effects in animal and human models of pain, co-administration of magnesium for post-operative epidural analgesia results in a reduction in fentanyl consumption without any side effects.

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PROFORMA

COMPARISION OF EPIDURAL BUPIVACAINE AND BUPIVACAINE-MAGNESIUM SULPHATE COMBINATION

NAME DATE

IP NO AGE SEX

HEIGHT WEIGHT

DIAGNOSIS SURGERY

PREANAESTHETIC EVALUATION

HISTORY

NIL ORAL FROM

PR BP

CVS RS OTHER SYSTEMS

AIRWAY ASA GRADE

ANAESTHESIOLOGIST SURGEON

INVESTIGATIONS

Hb URINE ALBUMIN

SUGAR

BLOOD SUGAR UREA CREATININE

CXR ECG

PRELOADING

GROUP I : BUPIVACAINE 0.5% 19ml at L2-L3 SPACE.

GROUP II : BUPIVACAINE 0.5% 19ml + 50 mg of MAGNESIUM SULPHATE

TIME	PR	BP	SPO2	LEVEL OF BLOCK	REMARKS

ONSET OF SENSORY BLOCK AT L3 FOR PINPRICK :

ONSET OF MOTOR BLOCKADE FOR ANKLE DORSIFLEXION:

NUMBER OF HYPOTENSION EPISODE:

TOTAL USAGE OF EPHEDRINE:

HIGHEST SEGMENT ACHIEVED:

TWO SEGMENT REGRESSION TIME:

SIDE EFFECTS: